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# Interleukin-10 protects against atherosclerosis by modulating multiple atherogenic macrophage function

Han X., Boisvert W.
*Kazan Federal University, 420008, Kremlevskaya 18, Kazan, Russia*

## Abstract

© Schattauer 2015. Atherosclerosis is primarily a disorder of lipid metabolism, but there is also a prominent chronic inflammatory component that drives the atherosclerotic lesion progression in the artery wall. During hyperlipidaemic conditions, there is a rapid influx of circulating monocytes into the atherosclerosis-prone areas of the arterial intima. These infiltrated monocytes differentiate into macrophages and take up the atherogenic lipoproteins in the intima of the vessel wall that have been modified within the lesion environment. Interleukin (IL)-10 is a prototypic anti-inflammatory cytokine made primarily by the macrophages and Th2 subtype T lymphocytes. In terms of atherosclerosis its major roles include inhibition of macrophage activation as well as inhibition of matrix metalloproteinase, pro-inflammatory cytokines and cyclooxygenase- 2 expression in lipid-loaded and activated macrophage foam cells. Recent discoveries suggest another important role of IL-10 in atherosclerosis: its ability to alter lipid metabolism in macrophages. The current review will highlight the present knowledge on multiple ways in which IL-10 mediates atherosclerosis. As macrophages play a critical role in all stages of atherosclerosis, the review will concentrate on how IL-10 regulates the activities of macrophages that are especially important in the development of atherosclerosis.

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